```
Connecting via Winsock to STN


Welcome to STN International!  Enter x:x

LOGINID:SSSPTA1623SQS

PASSWORD:
TERMINAL (ENTER 1, 2, 3, OR ?):2


* * * * * * * * * *   Welcome to STN International   * * * * * * * * * *

 NEWS  1           Web Page URLs for STN Seminar Schedule - N. America
 NEWS  2           "Ask CAS" for self-help around the clock
 NEWS  3  FEB 25   CA/CAPLUS - Russian Agency for Patents and Trademarks
                   (ROSPATENT) added to list of core patent offices covered
 NEWS  4  FEB 28   PATDPAFULL - New display fields provide for legal status
                   data from INPADOC
 NEWS  5  FEB 28   BABS - Current-awareness alerts (SDIs) available
 NEWS  6  FEB 28   MEDLINE/LMEDLINE reloaded
 NEWS  7  MAR 02   GBFULL: New full-text patent database on STN
 NEWS  8  MAR 03   REGISTRY/ZREGISTRY - Sequence annotations enhanced
 NEWS  9  MAR 03   MEDLINE file segment of TOXCENTER reloaded
 NEWS  10 MAR 22   KOREAPAT now updated monthly; patent information enhanced
 NEWS  11 MAR 22   Original IDE display format returns to REGISTRY/ZREGISTRY
 NEWS  12 MAR 22   PATDPASPC - New patent database available
 NEWS  13 MAR 22   REGISTRY/ZREGISTRY enhanced with experimental property tags

 NEWS EXPRESS  JANUARY 10 CURRENT WINDOWS VERSION IS V7.01a, CURRENT
               MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
               AND CURRENT DISCOVER FILE IS DATED 10 JANUARY 2005

 NEWS HOURS    STN Operating Hours Plus Help Desk Availability
 NEWS INTER    General Internet Information
 NEWS LOGIN    Welcome Banner and News Items
 NEWS PHONE    Direct Dial and Telecommunication Network Access to STN
 NEWS WWW      CAS World Wide Web Site (general information)

Enter NEWS followed by the item number or name to see news on that
specific topic.

  All use of STN is subject to the provisions of the STN Customer
  agreement.  Please note that this agreement limits use to scientific
  research.  Use for software development or design or implementation
  of commercial gateways or other similar uses is prohibited and may
  result in loss of user privileges and other penalties.

  * * * * * * * * * * * * * * STN  Columbus  * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 13:32:37 ON 28 MAR 2005

=> File Medline EMBASE Biosis CAplus
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                      ENTRY     SESSION
FULL ESTIMATED COST                                    0.21        0.21

FILE 'MEDLINE' ENTERED AT 13:32:44 ON 28 MAR 2005

FILE 'EMBASE' ENTERED AT 13:32:44 ON 28 MAR 2005
COPYRIGHT (C) 2005 Elsevier Inc. All rights reserved.
```

```
FILE 'BIOSIS' ENTERED AT 13:32:44 ON 28 MAR 2005
Copyright (c) 2005 The Thomson Corporation

FILE 'CAPLUS' ENTERED AT 13:32:44 ON 28 MAR 2005
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2005 AMERICAN CHEMICAL SOCIETY (ACS)

=> s myoD (3A) ((C or carboxy) (W) (terminal or terminus or end))
L1          13 MYOD (3A) ((C OR CARBOXY) (W) (TERMINAL OR TERMINUS OR END))

=> duplicate
ENTER REMOVE, IDENTIFY, ONLY, OR (?):remove
ENTER L# LIST OR (END):l1
DUPLICATE PREFERENCE IS 'MEDLINE, EMBASE, BIOSIS, CAPLUS'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L1
L2            4 DUPLICATE REMOVE L1 (9 DUPLICATES REMOVED)

=> file stnguide
COST IN U.S. DOLLARS                          SINCE FILE      TOTAL
                                                   ENTRY    SESSION
FULL ESTIMATED COST                               14.34      14.55

FILE 'STNGUIDE' ENTERED AT 13:34:27 ON 28 MAR 2005
USE IS SUBJECT TO THE TERMS OF YOUR CUSTOMER AGREEMENT
COPYRIGHT (C) 2005 AMERICAN CHEMICAL SOCIETY, JAPAN SCIENCE
AND TECHNOLOGY CORPORATION, AND FACHINFORMATIONSZENTRUM KARLSRUHE

FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Mar 25, 2005 (20050325/UP).

=> d l2 1-4 bib ab
YOU HAVE REQUESTED DATA FROM FILE 'MEDLINE, EMBASE' - CONTINUE? (Y)/N:y


L2   ANSWER 1 OF 4     MEDLINE on STN                    DUPLICATE 1
AN   2004283303     MEDLINE
DN   PubMed ID: 15183731
TI   A conserved enhancer element that drives FGF4 gene expression in the
     embryonic myotomes is synergistically activated by GATA and bHLH proteins.
AU   Iwahori Akiyo; Fraidenraich Diego; Basilico Claudio
CS   Department of Microbiology, New York University School of Medicine, New
     York, NY 10016, USA.
NC   CA078925 (NCI)
SO   Developmental biology, (2004 Jun 15) 270 (2) 525-37.
     Journal code: 0372762. ISSN: 0012-1606.
CY   United States
DT   Journal; Article; (JOURNAL ARTICLE)
LA   English
FS   Priority Journals
EM   200408
ED   Entered STN: 20040609
     Last Updated on STN: 20040813
     Entered Medline: 20040812
AB   FGF4 is the earliest member of the fibroblast growth factor (FGF) family
     expressed during embryogenesis where it plays essential roles in
     post-implantation development and limb growth and patterning.  The
     expression of the Fgf4 gene in specific developmental stages, including
     the ICM of the blastocyst, the myotomes, and the limb bud AER, is
     regulated by distinct enhancer elements (Hom) in the 3' UTR.  We
     previously identified the Hom3a region as the major DNA element
```

responsible for Fgf4 expression in the myotomes and AER, and showed that a conserved E-box is a target for the myogenic bHLH transcription factors MYF5 and MYOD. To further define the cis- and trans-acting elements that determine Hom3a activity, we conducted a mutational analysis of the ability of the Hom3a region to drive lacZ expression in the myotomes of transgenic mice. We identified a minimal enhancer of 226nt that contains four elements, including the E-box, necessary to drive gene expression in the myotomes. One of these elements is a binding site for the GATA family of transcription factors, and we show here that GATA 1-4 and 6 can synergize with MYF5 or MYOD to activate transcription of a reporter plasmid driven by a portion of the Hom3a enhancer including the GATA site and the E-box. In line with this finding, we could show a direct interaction between MYF5/MYOD and GATA-3 or GATA-4, mediated by the N-terminal and bHLH domains of MYF5/**MYOD** and the **C-terminal** zing finger domain of GATA-3/4. To further study the role of the Hom3a enhancer in directing Fgf4 expression and the function of FGF4 in limb and muscle development, we generated mutant mice in which the Fgf4 Hom3a region had been deleted (Delta3a). In situ hybridization analysis of sections from Delta3a/ Delta3a embryos at E11.5 showed a drastically reduced expression of Fgf4 mRNA in the myotomes and AER. However, these mice developed normally and show no limb or muscle defects, and the same was true of heterozygous mice in which one Fgf4 allele carried the Hom3a deletion and the other was a null allele (Delta3a/Fgf4(-)). Together, these results show that Hom3a is the major DNA enhancer element directing Fgf4 expression in myotomes and limb bud AER, and that its activity in the myotomes results at least in part from the synergistic action of GATA and bHLH myogenic factors that bind to evolutionary conserved sequences in the Hom3a enhancer. However, expression of Fgf4 in the myotomes or AER of murine embryos does not appear to be essential for muscle or limb development.

L2 ANSWER 2 OF 4 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
AN 2002364193 EMBASE
TI Tat-controlled protein acetylation.
AU Col E.; Gilquin B.; Caron C.; Khochbin S.
CS S. Khochbin, Lab. de Biol. Molec. et Cellulaire, Institut Albert Bonniot, Faculte de Medecine, Domaine de la Merci, 38706 La Tronche Cedex, France. khochbin@ujf-grenoble.fr
SO Journal of Biological Chemistry, (4 Oct 2002) 277/40 (37955-37960). Refs: 29
ISSN: 0021-9258 CODEN: JBCHA3
CY United States
DT Journal; Article
FS 004 Microbiology
LA English
SL English
AB Human immunodeficiency virus, type 1-encoded transactivator protein Tat is known to be a substrate of and to interact with several nuclear histone acetyltransferases (HATs). Here we show that Tat is a general inhibitor of histone acetylation by cellular HATs and that for at least one of them, the CREB-binding protein (CBP), it induces a substrate selectivity. Indeed, in the presence of Tat, the acetylation of histones by CBP was severely inhibited, while that of p53 and **MyoD** remained unaffected. The **C-terminal** domain of Tat, dispensable for the activation of viral transcription, was found to be necessary and sufficient to interfere with histone acetylation. These results demonstrate that Tat is able to selectively modulate cellular protein acetylation by nuclear HATs and therefore to take over this specific signaling system in cells.

L2 ANSWER 3 OF 4 MEDLINE on STN DUPLICATE 2
AN 2000069328 MEDLINE
DN PubMed ID: 10601020

TI Direct inhibition of G(1) cdk kinase activity by MyoD promotes myoblast cell cycle withdrawal and terminal differentiation.
AU Zhang J M; Zhao X; Wei Q; Paterson B M
CS Laboratory of Biochemistry, NCI, National Institutes of Health, Building 37 Room 4A21, 9000 Rockville Pike, Bethesda, MD 20892, USA.
SO EMBO journal, (1999 Dec 15) 18 (24) 6983-93.
Journal code: 8208664. ISSN: 0261-4189.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200001
ED Entered STN: 20000204
Last Updated on STN: 20000204
Entered Medline: 20000127
AB MyoD has been proposed to facilitate terminal myoblast differentiation by binding to and inhibiting phosphorylation of the retinoblastoma protein (pRb). Here we show that MyoD can interact with cyclin-dependent kinase 4 (cdk4) through a conserved 15 amino acid (aa) domain in the **C-terminus** of **MyoD**. **MyoD**, its **C-terminus** lacking the basic helix-loop-helix (bHLH) domain, or the 15 aa cdk4-binding domain all inhibit the cdk4-dependent phosphorylation of pRb in vitro. Cellular expression of full-length MyoD or fusion proteins containing either the C-terminus or just the 15 aa cdk4-binding domain of MyoD inhibit cell growth and pRb phosphorylation in vivo. The minimal cdk4-binding domain of MyoD fused to GFP can also induce differentiation of C2C12 muscle cells in growth medium. The defective myogenic phenotype in MyoD-negative BC3H1 cells can be rescued completely only when MyoD contains the cdk4-binding domain. We propose that a regulatory checkpoint in the terminal cell cycle arrest of the myoblast during differentiation involves the modulation of the cyclin D cdk-dependent phosphorylation of pRb through the opposing effects of cyclin D1 and MyoD.

L2 ANSWER 4 OF 4 MEDLINE on STN DUPLICATE 3
AN 1998352826 MEDLINE
DN PubMed ID: 9690516
TI p53 protein is activated during muscle differentiation and participates with MyoD in the transcription of muscle creatine kinase gene.
AU Tamir Y; Bengal E
CS Department of Biochemistry, Rappaport Institute for Research in the Medical Sciences, Faculty of Medicine, Technion-Israel Institute of Technology, Haifa.
SO Oncogene, (1998 Jul 23) 17 (3) 347-56.
Journal code: 8711562. ISSN: 0950-9232.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199808
ED Entered STN: 19980828
Last Updated on STN: 19980828
Entered Medline: 19980814
AB The p53 protein is a transcription factor involved in processes of cell growth and differentiation. The muscle creatine kinase (MCK) gene whose transcription is induced during muscle differentiation contains p53-binding sites. In this study we tested the involvement of p53 in the activation of MCK transcription during muscle differentiation of C2 cells. We have shown that the p53 protein is stabilized and its DNA binding and transcriptional activities are induced during muscle differentiation. At the stage of muscle-differentiation, p53 protein can induce the accurate transcription of a minimal p53-dependent MCK reporter gene. Moreover, p53 cooperates with MyoD in the induction of MCK transcription. The expression of a dominant negative p53 protein in muscle cells reduced the

expression of endogenous MCK gene. The dominant negative p53 protein abolished the cooperativity of wild type p53 with MyoD. Amino and **carboxy terminal** residues of **MyoD** required for the cooperation with p53 in transcription were identified. The cooperativity between the two proteins occurs also at the stage of DNA binding. We suggest that p53 protein is activated during myoblast differentiation and participates with MyoD in the induction of MCK transcription.

=> file stnguide

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.06	19.88

FILE 'STNGUIDE' ENTERED AT 13:34:59 ON 28 MAR 2005

Oncogene (1998) 17, 347-356

http://www.stockton-press.co.uk/onc

p53 protein is activated during muscle differentiation and participates with MyoD in the transcription of muscle creatine kinase gene

Yael Tamir and Eyal Bengal

Department of Biochemistry, Rappaport Institute for Research in the Medical Sciences, Faculty of Medicine, Technion-Israel Institute of Technology, Efron Street, P.O. Box 9649, Haifa 31096, Israel

The p53 protein is a transcription factor involved in processes of cell growth and differentiation. The muscle creatine kinase (MCK) gene whose transcription is induced during muscle differentiation contains p53-binding sites. In this study we tested the involvement of p53 in the activation of MCK transcription during muscle differentiation of C2 cells. We have shown that the p53 protein is stabilized and its DNA binding and transcriptional activities are induced during muscle differentiation. At the stage of muscle-differentiation, p53 protein can induce the accurate transcription of a minimal p53-dependent MCK reporter gene. Moreover, p53 cooperates with MyoD in the induction of MCK transcription. The expression of a dominant negative p53 protein in muscle cells reduced the expression of endogenous MCK gene. The dominant negative p53 protein abolished the cooperativity of wild type p53 with MyoD. Amino and carboxy terminal residues of MyoD required for the cooperation with p53 in transcription were identified. The cooperativity between the two proteins occurs also at the stage of DNA binding. We suggest that p53 protein is activated during myoblast differentiation and participates with MyoD in the induction of MCK transcription.

Keywords: p53; MyoD; cooperativity; muscle creatine kinase gene; muscle differentiation; transcriptional activation

Introduction

The p53 gene encodes a nuclear phospho-protein which functions as a tumor suppressor by negative regulation of cell cycle progression and the induction of apoptosis (Finlay *et al.*, 1989; Chen *et al.*, 1990; Gottlieb and Oren, 1996; Ko and Prives, 1996; Levine, 1997). p53 is also involved in the regulation of cell differentiation (Prokocimer and Rotter, 1994; Rotter *et al.*, 1994). Increased activity or expression of p53 protein was observed in processes such as hematopoiesis (Kastan *et al.*, 1991b; Shaulsky *et al.*, 1991; Aloni-Grinstein *et al.*, 1995) spermatogenesis (Almon *et al.*, 1993) and myogenesis (Soddu *et al.*, 1996).

The involvement of p53 in muscle differentiation is controversial. On the one hand, p53-null mice developed normally and exhibited normal muscle differentiation (Donehower *et al.*, 1992). Fibroblast cells, derived from p53-null mice were converted to muscle cells as a result of the ectopic expression of MyoD (Halevy *et al.*, 1995). On the other hand, interference with the endogenous wild type p53 protein in an established muscle cell line showed a striking reduction in the ability of these cells to differentiate (Soddu *et al.*, 1996). The apparent conflict may be explained if p53 is replaced by the presence of a redundant activity in the animal. The redundant function, however, cannot be activated in the context of already determined cell lines. In fact, a new gene, p73, with significant amino acid and functional similarities to p53 was recently identified (Jost *et al.*, 1997).

p53 protein functions as a transcription factor that binds to specific DNA sequences and activates gene transcription (Kern *et al.*, 1991; Farmer *et al.*, 1992). Between the genes that have been identified as specific targets for p53 protein are p21/WAF, MDM2, GADD45, cyclin G and bax, whose products function as regulators of cell growth (reviewed by Ko and Prives, 1996; Levine, 1997). p53 can activate the transcription of genes that are involved in differentiation – the κ light chain gene in B cell differentiation (Aloni-Grinstein *et al.*, 1995) and the muscle creatine kinase (MCK) gene in muscle differentiation (Weintraub *et al.*, 1991c).

The MCK gene is induced after myoblast cells have undergone cell-cycle arrest and during terminal differentiation. Two groups of muscle-specific transcription factors were shown to be involved in the transcription of MCK. The first is the MyoD family that is involved in the activation of many muscle-specific genes and therefore plays a pivotal role in driving the myogenic developmental process (reviewed in Weintraub *et al.*, 1991a). The second is the MEF2 family that is not specific to muscle but plays an important role in the transcription of many muscle-specific genes, including myogenin that belongs to the MyoD family. Members of the MyoD and MEF2 families activate MCK transcription by binding to nearby DNA sequences that are located in the enhancer region (reviewed in Olson *et al.*, 1995).

Weintraub and colleagues demonstrated in transient transfection experiments that p53 activated the transcription of MCK reporter gene (Weintraub *et al.*, 1991c). Activation was dependent on the presence of a sequence of 500 nucleotides found 2800 to 3300 base pairs upstream to the transcription start site. Subsequent studies identified a 50 bp fragment within this sequence (−3182 to −3133, relative to transcription start site) to which p53 could bind (Zambetti *et al.*, 1992). The 50 bp fragment was shown to act like an enhancer element in its ability to induce transcrip-

Correspondence: E Bengal
Received 12 November 1997; revised 26 February 1998; accepted 26 February 1998

Connecting via Winsock to STN

Welcome to STN International! Enter x:x

LOGINID:SSSPTA1623SQS

PASSWORD:
TERMINAL (ENTER 1, 2, 3, OR ?):2

* * * * * * * * * * Welcome to STN International * * * * * * * * * *

NEWS	1		Web Page URLs for STN Seminar Schedule - N. America
NEWS	2		"Ask CAS" for self-help around the clock
NEWS	3	FEB 25	CA/CAPLUS - Russian Agency for Patents and Trademarks (ROSPATENT) added to list of core patent offices covered
NEWS	4	FEB 28	PATDPAFULL - New display fields provide for legal status data from INPADOC
NEWS	5	FEB 28	BABS - Current-awareness alerts (SDIs) available
NEWS	6	FEB 28	MEDLINE/LMEDLINE reloaded
NEWS	7	MAR 02	GBFULL: New full-text patent database on STN
NEWS	8	MAR 03	REGISTRY/ZREGISTRY - Sequence annotations enhanced
NEWS	9	MAR 03	MEDLINE file segment of TOXCENTER reloaded
NEWS	10	MAR 22	KOREAPAT now updated monthly; patent information enhanced
NEWS	11	MAR 22	Original IDE display format returns to REGISTRY/ZREGISTRY
NEWS	12	MAR 22	PATDPASPC - New patent database available
NEWS	13	MAR 22	REGISTRY/ZREGISTRY enhanced with experimental property tags

NEWS EXPRESS	JANUARY 10 CURRENT WINDOWS VERSION IS V7.01a, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 10 JANUARY 2005
NEWS HOURS	STN Operating Hours Plus Help Desk Availability
NEWS INTER	General Internet Information
NEWS LOGIN	Welcome Banner and News Items
NEWS PHONE	Direct Dial and Telecommunication Network Access to STN
NEWS WWW	CAS World Wide Web Site (general information)

Enter NEWS followed by the item number or name to see news on that specific topic.

All use of STN is subject to the provisions of the STN Customer agreement. Please note that this agreement limits use to scientific research. Use for software development or design or implementation of commercial gateways or other similar uses is prohibited and may result in loss of user privileges and other penalties.

* * * * * * * * * * * * * STN Columbus * * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 12:40:06 ON 28 MAR 2005

=> File Medline EMBASE Biosis CAplus

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.21	0.21

FILE 'MEDLINE' ENTERED AT 12:40:13 ON 28 MAR 2005

FILE 'EMBASE' ENTERED AT 12:40:13 ON 28 MAR 2005

FILE 'BIOSIS' ENTERED AT 12:40:13 ON 28 MAR 2005
Copyright (c) 2005 The Thomson Corporation

FILE 'CAPLUS' ENTERED AT 12:40:13 ON 28 MAR 2005
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2005 AMERICAN CHEMICAL SOCIETY (ACS)

=>
=> s myoD (3A) (fragment or peptide)
L1 28 MYOD (3A) (FRAGMENT OR PEPTIDE)

=> duplicate
ENTER REMOVE, IDENTIFY, ONLY, OR (?):remove
ENTER L# LIST OR (END):l1
DUPLICATE PREFERENCE IS 'MEDLINE, EMBASE, BIOSIS, CAPLUS'
KEEP DUPLICATES FROM MORE THAN ONE FILE? Y/(N):n
PROCESSING COMPLETED FOR L1
L2 11 DUPLICATE REMOVE L1 (17 DUPLICATES REMOVED)

=> d l2 1-11 bib ab

L2 ANSWER 1 OF 11 MEDLINE on STN DUPLICATE 1
AN 2001673410 MEDLINE
DN PubMed ID: 11577095
TI Acetylation of MyoD by p300 requires more than its histone acetyltransferase domain.
AU Polesskaya A; Harel-Bellan A
CS CNRS UPR 9079, Institut Andre Lwoff, 7 Rue Guy Moquet, 94800 Villejuif, France.
SO Journal of biological chemistry, (2001 Nov 30) 276 (48) 44502-3. Electronic Publication: 2001-09-27. Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200201
ED Entered STN: 20011126
Last Updated on STN: 20030105
Entered Medline: 20020110
AB MyoD, an essential transcription factor involved in muscle cell terminal differentiation, is regulated by acetylation, as are a number of other transcription factors, but the histone acetyltransferase enzyme responsible for this acetylation is a matter of controversy. In particular, contradictory findings have been reported concerning the ability of CBP/p300 to acetylate MyoD in vitro. Here we provide an explanation for this discrepancy: although full-length p300 does indeed acetylate **MyoD**, a **fragment** of p300 corresponding to its histone acetyltransferase domain does not. In addition to clearly demonstrating that p300 acetylates MyoD in vitro, these results underscore the necessity of using full-length histone acetyltransferase enzymes to draw valid conclusions from acetylation experiments.

L2 ANSWER 2 OF 11 MEDLINE on STN
AN 2002036121 MEDLINE
DN PubMed ID: 11761848
TI Construction of eukaryotic expression plasmid for mouse myogenic regulatory factor MyoD gene.
AU Qin R F; Gu X M; Chen J W
CS Department of Maxillofacial Surgery, Qin-Du Stomatological College, Fourth Military Medical University, Xi'an Shanxi, P. R. China 710032.
SO Zhongguo xiu fu chong jian wai ke za zhi = Zhongguo xiufu chongjian waike

zazhi = Chinese journal of reparative and reconstructive surgery, (2001 Sep) 15 (5) 257-60.
Journal code: 9425194. ISSN: 1002-1892.
CY China
DT Journal; Article; (JOURNAL ARTICLE)
LA Chinese
FS Priority Journals
EM 200211
ED Entered STN: 20020124
Last Updated on STN: 20021211
Entered Medline: 20021122
AB OBJECTIVE: To construct eukaryotic expression plasmid of mouse myogenic regulatory factor MyoD gene for further study on MyoD gene function in molecular regulatory mechanism in skeletal muscle repair. METHODS: The plasmids PEMMBC2 beta 5 containing full cDNA length of MyoD inserted in EcoRI restriction site, were first propagated in Escherichia coli DH5a, then extracted and purified with the Wizard Plus Minipreps DNA Purification System (Promega, USA). The coding sequence of MyoD in PEMMBC2 beta 5 was confirmed by agarose gel electrophoresis and DNA sequence analysis. After plasmids PEMMBC2 beta 5 and plasmids pcDNA3-neo were prepared by digestion with EcoRI, the **MyoD** cDNA **fragment** was inserted into EcoRI site in pcDNA3-neo eukaryotic expression vector, and pcDNA3/MyoD was formed. The pcDNA3/MyoD, digested with restriction enzymes, was found to contain the MyoD cDNA sequence by agarose gel electrophoresis analysis. RESULTS: The extracted and purified PEMMBC2 beta 5 contained the correct nucleotide sequence for the full length of **MyoD** cDNA **fragment**. The **MyoD** cDNA **fragment** had been inserted into the eukaryotic expression plasmid pcDNA3-neo, which formed the pcDNA3/MyoD. CONCLUSION: The pcDNA3/MyoD, a eukaryotic expression plasmid, for MyoD is constructed successfully.

L2 ANSWER 3 OF 11 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1999:403151 CAPLUS
DN 131:209933
TI Partial sequence of the bovine (Bos taurus coreanae) myogenic factor encoding gene MyoD
AU Kim, H. S.; Park, E. W.; Yoon, D. H.; Kim, H. B.; Cheong, I. C.; Cho, B. W.; Im, K. S.
CS Division of Animal Breeding and Reproduction, National Livestock Research Institute, RDA, S. Korea
SO Asian-Australasian Journal of Animal Sciences (1999), 12(5), 689-694
CODEN: AJASEL; ISSN: 1011-2367
PB Asian-Australasian Association of Animal Production Societies
DT Journal
LA English
AB This experiment was carried out to isolate the partial bovine (Bos taurus coreanae) myogenic factor encoding gene, MyoD, using the rat myogenic factor (MyoD) gene sequence and to compare the gene sequence between another myogenic factor (Myf 5) and MyoD gene of the bovine. To make the probe and isolate the MyoD gene, PCR was performed to amplify rat and bovine MyoD gene including exon I, II and intron I. The homol. between mouse and bovine MyoD is high; bovine MyoD gene shows 17 different gene sequence region compared to rat MyoD. Among those, two regions have significant differences; one is the exon I part between 2834 and 2850 bp, the other is intron part between 3274 and 3303 bp of the mouse. At this region homol. was 40% in the former and 50% in the latter. Homol. between bovine MyoD and Myf5 was 83% in the exon 1. Especially exon I in the Myf5 602-617 bp and 651-683 bp have significant differences. These results suggest that MyoD gene have a similar gene structure in mouse and bovine and MyoD and Myf5 of the bovine, at least in part, have a similar expression and activity.
RE.CNT 19 THERE ARE 19 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 4 OF 11 MEDLINE on STN DUPLICATE 2
AN 1998001585 MEDLINE
DN PubMed ID: 9343420
TI The basic domain of myogenic basic helix-loop-helix (bHLH) proteins is the novel target for direct inhibition by another bHLH protein, Twist.
AU Hamamori Y; Wu H Y; Sartorelli V; Kedes L
CS Institute for Genetic Medicine and Department of Biochemistry and Molecular Biology, University of Southern California School of Medicine, Los Angeles 90033, USA.
SO Molecular and cellular biology, (1997 Nov) 17 (11) 6563-73.
Journal code: 8109087. ISSN: 0270-7306.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199711
ED Entered STN: 19971224
Last Updated on STN: 19971224
Entered Medline: 19971121
AB In vertebrates, the basic helix-loop-helix (bHLH) protein Twist may be involved in the negative regulation of cellular determination and in the differentiation of several lineages, including myogenesis, osteogenesis, and neurogenesis. Although it has been shown that mouse twist (M-Twist) (i) sequesters E proteins, thus preventing formation of myogenic E protein-MyoD complexes and (ii) inhibits the MEF2 transcription factor, a cofactor of myogenic bHLH proteins, overexpression of E proteins and MEF2 failed to rescue the inhibitory effects of M-Twist on MyoD. We report here that M-Twist physically interacts with the myogenic bHLH proteins in vitro and in vivo and that this interaction is required for the inhibition of MyoD by M-Twist. In contrast to the conventional HLH-HLH domain interaction formed in the MyoD/E12 heterodimer, this novel type of interaction uses the basic domains of the two proteins. While the MyoD HLH domain without the basic domain failed to interact with M-Twist, a **MyoD peptide** containing only the basic and helix 1 regions was sufficient to interact with M-Twist, suggesting that the basic domain contacts M-Twist. The replacement of three arginine residues by alanines in the M-Twist basic domain was sufficient to abolish both the binding and inhibition of MyoD by M-Twist, while the domain retained other M-Twist functions such as heterodimerization with an E protein and inhibition of MEF2 transactivation. These findings demonstrate that M-Twist interacts with MyoD through the basic domains, thereby inhibiting MyoD.

L2 ANSWER 5 OF 11 MEDLINE on STN DUPLICATE 3
AN 97398491 MEDLINE
DN PubMed ID: 9250861
TI Genetic variation in the porcine myogenin gene locus.
AU Soumillion A; Erkens J H; Lenstra J A; Rettenberger G; te Pas M F
CS DLO-Institute for Animal Science and Health (ID-DLO), P.O. Box 65, 8200 AB Lelystad, The Netherlands.
SO Mammalian genome : official journal of the International Mammalian Genome Society, (1997 Aug) 8 (8) 564-8.
Journal code: 9100916. ISSN: 0938-8990.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-X89007; GENBANK-X89209; GENBANK-X89210
EM 199709
ED Entered STN: 19970926
Last Updated on STN: 19970926
Entered Medline: 19970912
AB The myogenin (MYOG) gene fulfills a key function in muscle differentiation by controlling the onset of myoblast fusion and the establishment of

myofibers. In meat-producing animals like pigs and cattle, myofiber numbers have been related to growth capacity. We have characterized the porcine MYOG gene to detect genetic variation at this locus and to relate it to growth characteristics. MYOG gene fragments were isolated by PCR on genomic DNA and by screening a genomic library with a mixture of the four human **MyoD** cDNA **fragments**. Both the exons and promoter region were very similar to the human and mouse genes. Southern blot analysis of 105 unrelated pigs revealed three polymorphic MspI sites, located in the promoter region, the second intron, and at the 3' side of the gene. PCR-RFLP tests detecting four MYOG alleles were developed. PCR analysis of a panel of pig-rodent somatic cell hybrids confirmed the genetic localization of MYOG on pig Chromosome (Chr) 9. The PCR-RFLP tests and microsatellite markers on Chr 9 offer the possibility to genotype large numbers of pigs for studies of genetic linkage to meat deposition and growth characteristics.

L2 ANSWER 6 OF 11 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1998:31547 CAPLUS
DN 128:227573
TI The self-association of basic helix-loop-helix peptides
AU Wendt, H.; Thomas, R. M.
CS Department Biological Chemistry Molecular Pharmacology, Harvard Medical School, Boston, MA, 02115, USA
SO Progress in Colloid & Polymer Science (1997), 107, 115-121
CODEN: PCPSD7; ISSN: 0340-255X
PB Dr. Dietrich Steinkopff Verlag GmbH & Co. KG
DT Journal
LA English
AB As part of a study into the homo- and hetero-oligomerization properties of muscle-specific transcriptional factors, and their interaction with DNA, sedimentation equilibrium studies, accompanied by CD measurements, were made on peptides derived from the basic helix-loop-helix (bHLH) regions of MyoD and E47. In addition, a chimeric peptide, in which residues from the loop region of E47 were substituted into that of MyoD, a fluorescently labeled derivative of the **MyoD**-bHLH **peptide** and a disulfide crosslinked version of MyoD-bHLH were also investigated. MyoD-bHLH was found to form a monomer-tetramer equilibrium in the micromolar concentration range, while E47-bHLH exists as a highly associated dimer. The MyoD-bHLH derivs. appear to exhibit the same oligomerization behavior as their MyoD-bHLH parent. CD studies of the disulfide-crosslinked peptide show that a level of organization higher than that of the dimer is required for structural stability in the MyoD-bHLH system. The role of self-association in the context of the biol. function of these proteins is discussed.

L2 ANSWER 7 OF 11 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1995:592054 CAPLUS
DN 123:27141
TI Interactions of Myogenic bHLH Transcription Factors with Calcium-Binding Calmodulin and S100a (αα) Proteins
AU Baudier, Jacques; Bergeret, Evelyne; Bertacchi, Nathalie; Weintraub, Harold; Gagnon, Jean; Garin, Jerome
CS Departement de Biologie Moleculaire et Structurale, CEA, Grenoble, 38054, Fr.
SO Biochemistry (1995), 34(24), 7834-46
CODEN: BICHAW; ISSN: 0006-2960
PB American Chemical Society
DT Journal
LA English
AB MyoD belongs to a family of myogenic basic helix-loop-helix (bHLH) transcription factors that activate muscle-specific genes. The basic helix I sequence of the bHLH motif contains a consensus sequence for protein kinase C (PKC) substrates. We show here that MyoD is indeed phosphorylated by PKC in vitro on Thr 115 within the basic part of the

bHLH motif. By analogy with calmodulin-target peptide models, we also identified within the consensus basic helix I motif of myogenic proteins a conserved putative calmodulin/S100-binding domain. Calcium-dependent interaction between MyoD with calmodulin and the abundant muscle S100a(αα) proteins was demonstrated by affinity chromatog. and crosslinking expts. The binding of calmodulin and S100a inhibited MyoD phosphorylation by PKC as well as MyoD DNA binding activity. S100a was found to be more efficient than calmodulin in antagonizing DNA binding to MyoD. We next developped a rapid purification method for bacterial recombinant MyoD-bHLH domain by affinity chromatog. using a calmodulin-Sepharose column and investigated the phosphorylation of that peptide by PKC and its interactions with calmodulin and S100a. We confirmed the phosphorylation of the threonine residue 115 in the MyoD-bHLH by PKC with a Km of 0.8 μM. Calmodulin and S100a binding inhibited MyoD-bHLH phosphorylation by PKC. A strict calcium-dependent interaction between calcium binding proteins and the MyoD-bHLH was identified by native gel electrophoresis and fluorescence spectroscopy with 5-(dimethylamino)naphthalene-1-sulfonylcalmodulin. The MyoD-bHLH bound to fluorescently labeled 5-(dimethylamino)naphthalene-1-sulfonylcalmodulin with a dissociation constant around 20 nM. S100a inhibited stoichiometrically the binding of the bHLH peptide for labeled calmodulin, suggesting an affinity of S100a for the bHLH peptide at least 1 order of magnitude higher than calmodulin. In favor of an in vivo interaction between S100a and MyoD, we report that S100a- and MyoD-like immunoreactivities colocalize in H9c2 cells, and that a significant amount of MyoD-like immunoreactivity is recovered in the S100a immunoppt. from crude H9c2 cell extract in the presence of calcium. We propose that myogenic proteins represent a new family of calmodulin/S100-binding PKC substrates and that calmodulin/S100a could participate in the regulation of the bHLH myogenic protein activities.

L2 ANSWER 8 OF 11 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
AN 95152061 EMBASE
DN 1995152061
TI Two nuclear localization signals present in the basic-helix 1 domains of MyoD promote its active nuclear translocation and can function independently.
AU Vandromme M.; Cavadore J.-C.; Bonnieu A.; Froeschle A.; Lamb N.; Fernandez A.
CS CRBM, CNRS, INSERM, BP 5051,34033 Montpellier Cedex, France
SO Proceedings of the National Academy of Sciences of the United States of America, (1995) 92/10 (4646-4650).
ISSN: 0027-8424 CODEN: PNASA6
CY United States
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
AB MyoD, a member of the family of helix-loop-helix myogenic factors that plays a crucial role in skeletal muscle differentiation, is a nuclear phosphoprotein. Using microinjection of purified MyoD protein into rat fibroblasts, we show that the nuclear import of MyoD is a rapid and active process, being ATP and temperature dependent. Two nuclear localization signals (NLSs), one present in the basic region and the other in the helix 1 domain of MyoD protein, are demonstrated to be functional in promoting the active nuclear transport of **MyoD**. Synthetic **peptides** spanning these two NLSs and biochemically coupled to IgGs can promote the nuclear import of microinjected IgG conjugates in muscle and nonmuscle cells. Deletion analysis reveals that each sequence can function independently within the MyoD protein since concomittant deletion of both sequences is required to alter the nuclear import of this myogenic factor. In addition, the complete cytoplasmic retention of a β-galactosidase-MyoD fusion mutant protein, double deleted at these two NLSs, argues against the existence of another functional NLS motif in MyoD.

L2 ANSWER 9 OF 11 MEDLINE on STN DUPLICATE 4
AN 93003169 MEDLINE
DN PubMed ID: 1327135
TI Folding topology of the disulfide-bonded dimeric DNA-binding domain of the myogenic determination factor MyoD.
AU Starovasnik M A; Blackwell T K; Laue T M; Weintraub H; Klevit R E
CS Department of Biochemistry, University of Washington, Seattle 98195.
SO Biochemistry, (1992 Oct 20) 31 (41) 9891-903.
Journal code: 0370623. ISSN: 0006-2960.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199211
ED Entered STN: 19930122
Last Updated on STN: 19930122
Entered Medline: 19921118
AB The myogenic determination factor MyoD is a member of the basic-helix-loop-helix (bHLH) protein family. A 68-residue **fragment** of **MyoD** encompassing the entire bHLH region (MyoD-bHLH) is sufficient for protein dimerization, sequence-specific DNA binding in vitro, and conversion of fibroblasts into muscle cells. The circular dichroism spectrum of MyoD-bHLH indicates the presence of significant alpha-helical secondary structure; however, the NMR spectrum lacks features of a well-defined tertiary structure. There is a naturally occurring cysteine at residue 135 in mouse MyoD that when oxidized to a disulfide induces MyoD-bHLH to form a symmetric homodimer with a defined tertiary structure as judged by sedimentation equilibrium ultracentrifugation and NMR spectroscopy. Oxidized MyoD-bHLH retains sequence-specific DNA-binding activity, albeit with an apparent 100-1000-fold decrease in affinity. Here, we report the structural characterization of the oxidized MyoD-bHLH homodimer by NMR spectroscopy. Our findings indicate that the basic region is unstructured and flexible, while the HLH region consists of two alpha-helices of unequal length connected by an as yet undetermined loop structure. Qualitative examination of interhelical NOEs suggests several potential arrangements for the two helix 1/helix 2 pairs in the symmetric oxidized dimer. These arrangements were evaluated for whether they could incorporate the disulfide bond, satisfy loop length constraints, and juxtapose the two basic regions. Only a model that aligns helix 1 parallel to helix 1' and antiparallel to helix 2 was consistent with all constraints. Thus, an antiparallel four-helix bundle topology is proposed for the symmetric dimer. This topology is hypothesized to serve as a general model for other bHLH protein domains.

L2 ANSWER 10 OF 11 MEDLINE on STN DUPLICATE 5
AN 92279026 MEDLINE
DN PubMed ID: 1594450
TI The human M creatine kinase gene enhancer contains multiple functional interacting domains.
AU Trask R V; Koster J C; Ritchie M E; Billadello J J
CS Department of Medicine, Washington University School of Medicine, St Louis, MO 63110.
NC HL-38868 (NHLBI)
SO Nucleic acids research, (1992 May 11) 20 (9) 2313-20.
Journal code: 0411011. ISSN: 0305-1048.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199206
ED Entered STN: 19920710
Last Updated on STN: 19920710

Entered Medline: 19920630

AB Cis-elements (-933 to -641) upstream of the human M creatine kinase gene cap site contain an enhancer that confers developmental and tissue-specific expression to the chloramphenicol acetyltransferase gene in C2C12 myogenic cells transfected in culture. Division of the enhancer at -770 into a 5' **fragment** that includes the **MyoD** binding sites (-933 to -770) and a 3' fragment that includes the MEF-2 binding site (-770 to -641) resulted in two subfragments that showed minimal activity but in combination interacted in a position- and orientation-independent fashion to enhance activity of the SV40 promoter in transient transfection experiments. A 5' enhancer construct (-877 to -832) including only one (the low affinity) MyoD binding site was active when present in multiple copies. In contrast, a 3' enhancer construct (-749 to -732) including the MEF-2 binding site was inactive even when present in multiple copies. However, if the 5' construct was extended to include the high-affinity MyoD binding site (-877 to -803) the 5' and 3' constructs interacted in a position- and orientation-independent fashion to activate the SV40 promoter. Thus, the human M creatine kinase enhancer comprises multiple functional interacting domains.

L2 ANSWER 11 OF 11 MEDLINE on STN DUPLICATE 6.
AN 92339895 MEDLINE
DN PubMed ID: 1321778
TI Isolation and structural analysis of the rat MyoD gene.
AU Vaidya T B; Rhodes S J; Moore J L; Sherman D A; Konieczny S F; Taparowsky E J
CS Department of Biological Sciences, Purdue University, West Lafayette, IN 47907.
SO Gene, (1992 Jul 15) 116 (2) 223-30.
Journal code: 7706761. ISSN: 0378-1119.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-M74890; GENBANK-M74891; GENBANK-M74892; GENBANK-M79309; GENBANK-M79310; GENBANK-M84176; GENBANK-S38798; GENBANK-S38799; GENBANK-S40727; GENBANK-S40728
EM 199208
ED Entered STN: 19920911
Last Updated on STN: 19920911
Entered Medline: 19920825

AB We have cloned and determined the nucleotide (nt) sequence of a 6.5-kb genomic DNA **fragment** containing the rat **MyoD** gene (encoding a muscle regulatory factor, MyoD). Mouse fibroblasts transfected with this DNA display a high degree of conversion to a muscle phenotype, suggesting that this genomic clone contains sufficient sequence information to allow the production of the rat MyoD protein in these cells. The 6.5-kb genomic fragment contains the complete coding region of MyoD, distributed over three exons, plus 2.3 kb of 5'-noncoding sequence and 1.4 kb of 3'-noncoding sequence. Based on RNase protection assays, the major transcription start point of MyoD is located 210 nt 5' to a methionine start codon and 26 nt 3' to a TAAATA motif which bears similarity to a consensus recognition sequence (TATA) utilized by eukaryotic RNA polymerase II transcription complexes. The high degree of identity between the amino acid sequence of rat MyoD and the MyoD proteins isolated from other vertebrates indicates that this muscle regulatory protein has been evolutionarily conserved.

=> file stnguide

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	21.01	21.22

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-2.19	-2.19

FILE 'STNGUIDE' ENTERED AT 12:41:17 ON 28 MAR 2005

FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Mar 25, 2005 (20050325/UP).